Editorials

The Practice of Medicine Is Changing—or Is It?

THESE ARE TIMES for physicians to reflect upon why we came into medicine in the first place and perhaps to consciously rededicate ourselves to the ideas and ideals that inspired us in our earlier years when, for most of us, things were very different. While the money they thought they might earn may have motivated some, there must have been easier ways for them to get it. Most of those who were merely after money were smart enough to have made as much or more of it in other ways with a lot less demand on their talent and time. Money has always been important, of course, but physicians generally have been able to live comfortably and to practice conscientiously without thinking very much about money. But all this has changed. There is now good reason to believe that money will soon affect almost every decision in medical practice and almost every aspect of patient care, and this through no fault of practicing physicians or their patients.

The heart of medical practice is a physician, a patient and what takes place when the one seeks help and the other tries to help, using knowledge, skills, care and understanding that have been learned through training and experience. The patients who seek help have not changed very much, and the young men and women entering medical schools today have very much the same desire to help their fellowman and the same fascination with unfolding biomedical science that motivated most of us to join this profession in years past. The heart of medical practice has not changed, and the heart in it is still there, still warmly rewarding physicians who are able to relieve suffering, anxiety or pain in the patients who seek their help. These are times of almost unprecedented stress for physicians trying to practice good medicine, but each of us can gain the inner strength that is needed by making a conscious rededication to the ideas and ideals that brought us to this noble profession in the first place. Perhaps more of us should do this at this troubled time. We can also take genuine comfort in the reality that the more the practice of medicine changes, the more it remains the same.

MSMW

Reye's Syndrome—Still an Elusive Entity

REYE'S SYNDROME, first clearly described in 1963,[1] continues to be an elusive entity in 1984. Ideas concerning its etiology have come almost full circle from Mortimer and Lepow's early report of "varicella with hypoglycemia possibly due to salicylates"[2] to the recent epidemiologic evidence linking Reye's syndrome with salicylate use. Is the disorder merely a form of aspirin poisoning? The answer probably is not as simple as that. More likely, genetically determined susceptibility plus a complex interaction between toxicity of certain viruses and salicylate effects lead to the damage to mitochondria that underlies Reye's syndrome. A transient decrease in activity of multiple mitochondrial enzymes in turn explains all of the major clinical and laboratory findings including hyperammonemia (due to inhibition of the two mitochondrial enzymes of the Krebs urea cycle), fatty acidemia (due to interference with mitochondrial fatty acid oxidation), lactic acidemia (due to decreased activity of pyruvate carboxylase), accumulation of biogenic amines and aminoacidemia. Serum levels of several metabolites, including ammonia and lactate, correlate well with the severity of the central nervous system dysfunction.[3,4] The central nervous system toxicity of some, such as ammonia and short-chain fatty acids, is now well understood. Even the cerebral edema that frequently complicates Reye's syndrome can be explained on the basis of mitochondrial damage. To a large extent, it appears to be myelin edema, the same type seen experimentally in response to mitochondrial poisons such as triethyltin.[5]

The treatment of Reye's syndrome continues to be a controversial subject. The "therapeutic delirium" pointed out by Nadler in 1974[6] persists today. Many patients recover spontaneously without any therapy other than the intravenous administration of hydration and glucose. Almost everyone agrees that treatment of children who are awake should be limited to such simple measures. The approach to a comatose child varies greatly. Invasive measures including the placement of intracranial pressure monitors, administration of curare and artificial ventilation, hypothermia, barbiturate coma and exchange blood transfusion are used in various combinations and for various indications. Many experts apply such measures to all comatose children who have Reye's syndrome. Yet it is likely that many of these children would recover with less invasive approaches. Patients in whom metabolic variables such as blood ammonia and lactate levels are only mildly elevated early in the course appear to do well no matter what treatment is used,[3,4] provided that they are not injured by the therapeutic attempts. The tragedy of survival with severe, persistent brain damage, rare during the early days of treating Reye's syndrome, is now a relatively common occurrence with up to 20% of patients falling into this category in some series.[7] Injudicious use of hyperosmolar agents for control of cerebral edema is another possible cause of injury. Hyperosmolar coma, cerebral hemorrhage and disturbances of blood-brain barrier functions may result from such agents. Yet, they may be lifesaving in